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TB, or not TB: the New World Question.

Rob Rost

... tuberculosis was present well into antiquity in both the Old and New World
(Kelley and Micozzi 1984:381)

Archaeology is a discipline which tries to learn about humankind's prehistoric, and historic past. There are many variables that contribute to the "defining" of a once living population: Subsistence patterns and a populations' environmental setting, are good examples of this. In essence, the archaeologist is trying to reconstruct those variables which constitute a cultural tradition in a once living human population.

Disease is certainly one important variable or dimension influencing humankind's development. In particular, Tuberculosis (TB) has had major social ramifications for cultures in both the New World and the Old World. The hypothesis that TB had social ramifications is augmented when the relationship between *Mycobacteria Tuberculosis* and humans is viewed as a predator to prey relationship (McGrath 1988:324). The predator being *M. tuberculosis* and *Homo sapiens* being the prey. This relationship between *M. tuberculosis* and humans was present in the New World prior to Contact, that is before 1492. By looking at skeletal pathology of TB, the evidence for TB, and the paleopathology of TB in the New World, it will be argued that some of the pre–Historic Nations of the Americas were exposed to TB. There are two important questions that directly pertain to the discussion of TB in the New World: Is the *M. Tuberculosis* found in the New World the same as the *M. Tuberculosis* from the Old World?, and is prehistoric TB the same as the contemporary form of TB?

THE DYNAMICS OF MODERN TUBERCULOSIS

Any disease that involves the circulatory system, either in oxygen, mineral, or the manufacturing of blood can affect the bones of the human body (St. Hoyme, 1969:298). In discussion of the dynamics of TB, it is clear that TB is no exception and that chronic TB can lead to bony lesions in the skeleton. The causitive agent of the infectious disease TB is *Mycobacterium tuberculosis* (*M. tuberculosis*). *M. tuberculosis* has five strands: murine, avian, and piscine, human and bovine (El–Najjar 1981:86). Two strands of *M. tuberculosis*, *M. t. bovine* and *M. t. human*, are pathogenic to humans. When discussing *M. tuberculosis* in this paper I will be referring to these specific strands.

Transmission

The pathogen is usually transmitted by the dispersal of small, wet drops which contain a few bacilli. When these droplets reach an air cell of the lung, the alveoli, the bacilli are ingested by the alveolar macrophages. It is here in the lung where the primary focus of infection occurs, and is called the Ghonfocus. Next, the bacilli are carried to the lymph nodes at the hilum of the lung. In combination with the Ghon focus is called the primary complex (Clark et al., 1987:49; Ortner and Putschar, 1981:141). Much less common is the intestinal pathway with formation of a primary complex in the intestinal wall and mesenteric lymph nodes (Ortner and Putschar, 1981:141).

Immune Response (The Primary Complex)

The body's immune system reacts to these foreign pathogens, and most of the bacilli can be either destroyed or inhibited after a duration of two weeks by the cell–mediated immunity. In most individuals the primary complex has been resolved by the immune system and can either leave a fibrotic or calcified scar (Clark et al., 1987:49) However, in others, especially infants, one of the extra–pulmonary nucleuses may progress into a very dangerous disease, such as meningitis tuberculosis.

Infection

If the primary immunity fails, the bacilli get into the blood stream and set up a new infection in the joints, hip, and spinal column, causing permanent deformity (Burnet and White 1972:215; Ortner and Putschar, 1981:141). Both the primary complex, and the severity of the early haematogenous dissemination are dependent on the number of organisms and the immunological capacity of the patient. In addition, an individual who has just suffered through the primary complex could still have some living tubercle bacilli in the primary infection area. These living bacilli may go dormant, and may not reappear until later in adult life. The dormant bacilli might reappear again if the immune system of the individual becomes languid. The immunological capacity of an individual could be depreciated by malnutrition, other diseases, and/or trauma (Ortner and Putschar, 1981:141; Clark et al., 1987:49).

When the immune system can not effectively destroy or inhibit tubercle bacilli during the primary complex, *M. tuberculosis* can become an acute disease. However, when the bacilli reappear, the infection takes a longer time to develop because of the previously sensitized immune system (Middlebrook 1963:504). In other words, TB can also be a chronic disease.

This discussion on the dynamics of TB is based on relatively contemporary clinical cases, and comparison with past populations should be made with caution. However, the above framework still gives archaeologists a starting point for the diagnosis and identification of possible mechanisms (malnutrition, for example) responsible for the advent of TB in skeletal samples. With a basic understanding of the dynamics of TB, the next step is to look at how archaeologists are able to identify TB from skeletal remains.

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SKELETAL PATHOLOGY OF TUBERCULOSIS

> *... tuberculosis exhibits a relatively distinctive skeletal pattern of involvement which is more easily identifiable than many other diseases affecting osseous tissue*
>
> (Palkovich 1981:161).

When reviewing the literature, it is useful to remember that phrase "*relatively* distinctive skeletal pattern". It is a caution to archaeologists who are working with what they think are TB-infected skeletal remains, because TB can be misdiagnosed for other diseases (Pfeiffer, 1984:188; Morse, 1961, 1967; Rogers and Waldron, 1989; Kelley and El–Najjar, 1980; Alland, 1970:96–98). The possibility of misdiagnosis is magnified when dealing with disarticulated, poorly preserved, and/or fragmentary skeletal remains.

Diagnosis using the spine

Fragmentary remains are a particular problem when the spine is not present, because the traditional method of diagnosing TB is based on the spine (Morse 1967:249–250; Kelley and El–Najjar, 1980:166). The identifying characteristic of vertebral TB is seen on a few vertebrae with little or no regeneration of the bone lesion. The vertebral bodies can show resorptive lesions in the spinal column (Larsen, 1987:382), but osseous infections usually do not involve the neural arches, transverse and spinous processes. The vertebrae involved when the disease is in its advancing stages show decalcification and erosion. Body weight pressure is sufficient to cause an anterior collapse of the vertebrae, resulting in angular kyphosis (Pott's disease of the spine). This gives the individual a characteristic deformity (Morse, 1967:249–250; Rogers and Waldron, 1989:614).

Possible sources of confusion

Even when we have a vertebral column, however, there are various diseases and other pathological conditions with which TB can be confused. These are malignant tumours, fractures, pyogenic osteomyelitis, and fungal infections (Morse, 1961:Table 2, Pfeiffer, 1984:188). Tumours and fractures are not as problematic for archaeologists as pyogenic osteomyelitis and fungal infections. Malignant tumours can be differentiated by the diffuse vertebral involvement, which is most common in individuals older than 50 years of age (Kelley and El–Najjar, 1980:161). Differentiation between TB and fractures is made on the basis that fractures manifest: 1) marginal new bone growth; 2) no detectable osteolytic lesions; and 3) a maintainance of intervertebral disc integrity (Kelley and El–Najjar, 1980:161).

Osteomyelitis and Blastomycosis

The most difficult pathological differentiation is between TB and pyogenic osteomyelitis, and/or Blastomycosis (Ortner and Putschar, 1981:163; Moller–Christensen, 1983:133). The possibility of misdiagnosing is high for Pyogenic osteomyelitis because the organisms mimic TB in their location and appearance in skeletal material (Kelley and El–Najjar, 1980:162). Osteomyelitis is most commonly caused by *Staphylococcus aureus*, and can be disseminated through the blood stream. The bones and bone marrow become infected and when they are, osteomyelitis is the end result (Rogers and Waldron, 1989:612). Osteomyelitis occurs predominantly in adult skeletons, and similar to TB, it starts in the vicinity of the most rapidly growing parts of the bone–the growth plates (Ortner and Putschar, 1981:144). A diagnostic feature of pyogenic osteomyelitis is the formation of sinuses (cloacae) through which pus empties from the infected bone and bone marrow (Rogers and Waldron, 1989:612).

The earliest bone transformations are localized within the vertebral bodies, and differentiation from TB at this stage is virtually impossible. The confusion between TB and osteomyelitisis is easy to make because osteomyelitisis also affects the spine, especially in the lumbar and lower thoracic regions. The upper thoracic and cervical vertebrae are rarely involved. In addition, the vertebrae may collapse as infection spreads, and with long-standing infections the bones may become considerably swollen (Rogers and Waldron, 1989:612).

Osteomyelitis: The Effigy Mound Tradition

The problem of differentiating between TB and osteomyelitis can be best illustrated by giving an archaeological example. The Raisbeck Mounds are located in Wisconsin, and have been assigned to the relative date of the Middle phase of the Effigy Mound Tradition, A.D. 1100 (Sullivan, 1985:71). The individual under discussion is represented by one thoracic vertebra, one lumbar vertebra, a sacrum, fragments of the right and left scapulae, capitus humerus, and a left femur. There is a great deal of destruction of the spongy bone in the centrum of the lumbar. The pathology has affected the posterior and inferior aspects of the vertebral body while the superior and anterior surfaces of the centrum remained unaffected (Sullivan, 1985:71). Although it appears that the above diagnosis may suggest osteomyelitis, Sullivan (1985:73) eliminates osteomyelitis based on the observation that the expected reactive bone formation is absent from the lesion. He (1985:74) concludes that it is impossible to distinguish between TB and blastomycosis, but he favours the former as causing the pathology. I offer the Effigy Mound Tradition as an example because I think it reflects the importance of not giving a skeletal diagnosis quickly or dogmatically.

Other markers of Osteomyelitis

There are other skeletal pathological markers that differentiate between these diseases. First, with the exception of children, the bony reactions are usually only present in skulls when osteomyelitis is the causitive agent (Ortner and Putschar, 1981:163). Second, in osteomyelitis there is anterior new bone growth and bridging of vertebrae. Third, there are abscesses in the vertebral body surrounded by bony margins when the pathology is caused by osteomyelitis. Fourth, there is an association between large sequestra, and new subperiosteal bone growth, accompanied with sinus tracts *cum* osteomyelitis. In contrast, there are small or a lack of sequestra generally associated with TB infected bones (Kelley and El–Najjar, 1980:166).

Blastomycosis

Blastomycosis (fungal infection) is caused by *Blastomyces dermatitidis* which predominantly enters the body through the respiratory tract (Ortner and Putschar, 1981:224). Its main distribution is in North America, predominately in Eastern Canada and Eastern United States. The ratio in disseminated cases is 5:1 for male and female, respectively. The focal points of infection are the vertebrae (vertebral collapse is rare), skull, tibia, and tarsus. In long bones the foci are the sub–articular epiphysial and metaphysial areas (Ortner and Putschar, 1981:224).

Blastomycosis: The Averbuch site

The possibility of misdiagnosing a lesion as TB instead of Blastomycosis in a skeleton is always present. Take, for example, the Averbuch site in Tennessee (A.D 1275). Kelley and Eisenberg (1987:92) state that the most frequently observed lesions occurred in the spine, ribs, tibiae, and pelvis in descending order. The bio–culture settings at Averbuch contain numerous elements conducive to the dispersal of TB. It was concluded on the basis of lesion appearance, location, distribution, as well as sex and age distribution, that both diseases were present, with blastomycosis predominating (1987:97–98).

Kelley and Eisenberg (1987) present a convincing case for Blastomycosis as being the main agent of skeletal infection. However, I think that in this case, Kelley and Eisenberg are overemphasizing the importance of demographic patterning, and more specifically the importance of sex distribution. The difficulties in constructing demographic patterning have been outlined by Wood and colleagues in a relatively recent article (Wood et al., 1987). I am not criticizing the importance of assessing sex and the possible correlation to disease per se, but the way in which Kelley and Eisenberg present evidence in their article.

Blastomycosis: The assessment of sex-bias

In the case of modern studies, Kelley and Eisenberg (1987:95) state that the sex bias is partially due to the fact that males are in closer contact with soil more frequently. They might be farmers, for example. I agree that this hypothesis is possible in a modern context. However, this hypothesis is supposed to explain the sex bias in the Averbuch sample. The problem with this is that women predominately tilled the soil while men hunted in most prehistoric populations in the New World. The afflicted ratio for male to female, however, is nearly 2 to 1 (Kelley and Eisenberg, 1987:96). I would suspect that there would be a higher frequency of females in the Averbuch sample if Blastomycosis was the disease. There are two possibilities which account for this: 1) tuberculosis is the only disease affecting the skeletal material; and/or 2) women have a greater resistance to fungal infections. Kelley and Eisenberg do mention the latter possibility (1987:95), but I think they do not elaborate enough on this important area. Hence, as mentioned earlier, the greater exposure to soils hypothesis becomes the prominent explanation for the male to female ratio.

Finally, Kelley and Eisenberg (1987) do not address the issue that Blastomycosis is not transmitted from person to person, and consequently, a low frequency of infection is expected in the skeletal population (Pfeiffer, 1986:29). The only skeletal data that the article gives is that out of 766 burials utilized, 47 exhibited lesions that resembled TB or Blastomycosis (Kelley and Eisenberg, 1987:92). Therefore, approximately six percent of the skeletal population was infected by "tuberculosis–like" lesions. Unfortunately, there are no comparative pathologies given (trauma, and other diseases, for example) to indicate the relative frequency in the population. If there is a relatively high frequency of "tuberculosis–like" lesions then I would argue that TB is the prominent agent of infection. Conversely, Blastomycosis would be the most probable infecting disease if there is a low frequency of affected skeletal material. The above example should serve to illustrate that the diagnosis of TB is not always, if ever, an "either/or" (Kelley and Eisenberg, 1987:98) conclusion.

DIAGNOSIS OVERVIEW

It appears, then, that the differentiation between TB, pyogenic osteomyelitis, and Blastomycosis is problematic and far from being straightforward. Even with contemporary technology, I think that archaeologists have to realize that in some cases it is almost impossible to unequivocally diagnose TB in skeletal material. However, I think that if an archaeologist utilizes a multi–variant approach to the diagnosis of TB and through the process of elimination a sound case can be presented for or against TB.

First and foremost, there is the problem of determining the nature of the pathological bone changes and mapping their distribution (Rogers and Waldron, 1989:622). The importance of mapping the lesions is that the end result is a pattern of the disease. For example, TB occurs frequently in the head of femur, hip, knee, articular surfaces of joints, lumbar, and thoracic vertebrae (Long and Merbes 1981:80, Kelley 1989:197, Kelley and Eisenberg 1987:93, Davidson and Horowitz, 1970:78–79; see Ortner and Putschar, 1981). According to Kelley and Micozzi (1984:381), the diagnosis that has been described above is the traditional method of looking at secondary skeletal lesions. However, approximately ninety percent of human TB is in the form of chronic pulmonary disease (Kelley and Micozzi 1984:381). Hence, Kelley and Micozzi suggest a new diagnostic approach by looking for lesions on the internal aspect of the ribs. The most common symptom is diffuse periostitis, but there can also be localized abscesses which appear to correlate to areas that are infected by chronic pulmonary infection (Kelley and Micozzi 1984:381). In short, although numerous diseases are known to produce lesion similar to TB, only a few possess a similar lesion pattern, for example rib–sternum (Kelley and El–Najjar, 1980:167).

Second, there has to be a detailed reconstruction of the physical and cultural environment (Kelley and Eisenberg, 1987:90; Goodman, 1993) which could eliminate the possibility of certain diseases. For example, blastomycosis is predominately found in Eastern North America. With a note of caution about using this criterion, Blastomycosis could possibly be eliminated as a cause of "tuberculosis–like" lesions in a once living population through geographic default.

Keeping the problems and shortcomings of identifying TB in archaeological populations in mind, it is now possible to critically discuss the evidence for TB and the paleopathology of TB in the New World.

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TB, OR NOT TB: THE NEW WORLD EVIDENCE

Cultural Artifacts

I think that the best evidence of TB in the New World is derived from the individuals themselves (i.e. the skeletal material and mummies). Some researchers, on the other hand, have suggested that Native art, and other cultural material provides possible proof for the existence of TB in prehistoric America (Dubos and Dubos, 1952:5; Sigerist, 1962:206). The prehistoric cultural materials consist of clay figurines, pictographs and anthropomorphic water bottles who appear to be hunchbacked.

The anthropomorphic water bottles are from the Inca tradition, and supposedly show characteristics of pigeon breast deformity. It is supposed that they represent victims of TB (Lichtor and Lichtor 1957:1398; Ritchie 1952:309). In addition, some of the prehistoric Cayuga pottery pipes have been interpreted as representing a kyphotic spine (a hallmark of TB) and pigeon breasted individuals (Wells, 1966:100).

The pictographs that are interpreted as tuberculosis induced spinal deformities occur throughout the Southwestern United States (Morse, 1961:495). The individuals depicted in these pictographs are mostly lying on their backs, and holding an object that looks like a flute. The flutes have been interpreted as occupational therapy for the afflicted individual (Morse, 1961:495). There is also a Monte Alban dancer that has been put forward to support the notion that art reflects the presence of TB in the New World (Morse, 1961:495).

In light of the possibility for overlapping bone transformations from independent diseases, I think that the interpretation of a piece of art as representative of disease, let alone a single disease, is too presumptuous and circumstantial. The anthropomorphic water bottles and figurines, could be the result of the artists desire to create symmetry (1967:253). They also could have been the duplication of a person that was bending over (Morse 1967:253), or simply art for art's sake.

Skeletal Remains

The most convincing evidence for the presence of tuberculosis in the pre-Contact New World is from the numerous archaeological reports of prehistoric sites and their skeletal remains. These skeletal remains have lesions that are consistent with the diagnosis of tuberculosis (Larsen 1987:382).

The first ground breaking piece of evidence for the existence of TB in the Americas was the discovery of a mummy bundle burial (Allison et al., 1973:985). This mummy was found in an undisturbed grave in Southern Peru, and dates back to approximately the eighth century A.D. The mummy was a paralysed male aged between 8–10 years old, and his lumbar region was kyphotic. Meticulous examination of the lower lobe of the right lung revealed small white nodules resembling tubercles. Similar lesions were found in the kidney and the liver (Allison et al., 1973:985). Many of these lesions had acid–fast bacilli, with the greatest amount being in the lungs. It was concluded that primary complex tuberculosis was present, and was the cause of death for the boy (Allison et al., 1973:990). According to Sievers and Fisher (1981:234), this mummy is the solution to the dilemma of TB being present in the Americas prior to 1492. I agree with this assessment, in light of all the other archaeological evidence.

For example, there are three Iroquoian skeletons from New York thought to have suffered from TB (Ritchie 1952:309). There is also a pre–historic human spine from from an Indian burial ground in Tennessee exhibiting the signs of a slow healing of TB in the thoracolumbar region (Lichtor and Lichtor 1957:1398). There are a number of other examples of pre-Contact sites (some proto–historic) with TB-affected skeletons: Uxbridge, Ontario (Pfeiffer, 1983, 1984); Mobridge site, South Dakota (Kelley and Eisenberg, 1987); and the Pueblo site of Kechipawan (Lahr and Bowman, 1992).

Moreover, Buikstra (1981:9) suggests that there is a low record of TB in current skeletal collections from the Americas because of sample biases made by previous archaeologists. The bias in question is the result of previous archaeologists presenting prehistoric cases of TB on basis of the extreme examples of this disease: the ninety degree kyphosis and extensive ankylosis. Extra-vertebral involvement was not commonly considered (Buikstra 1981:9).

The question now for archaeologists is not whether TB was present, but rather how it occurred in the New World (Kelley and Micozzi 1984:385).

THE PALEOPATHOLOGY OF TUBERCULOSIS IN THE NEW WORLD

Thus far, I have presented archaeological evidence which strongly supports the notion that TB existed in the Americas before 1492. Nonetheless, the presence of TB in the New World prior to Contact does present a theoretical problem. If there were no cattle in the prehistoric Americas (Lovell, 1987:53), how could *M. t. Human* mutate from *M. t. bovis*? On the basis of Old World archaeological and written evidence, most researchers advocate the theory that *M. Human* derived from *M. bovis*, and this occurred shortly after the domestication of cattle and urbanisation (Morse, 1961; Manchester, 1983:39; Wells 1966:98; Polgar, 1967:205; Hare 1967:127; Burnet and White 1973:214; Murdochand Gray 1974:132; Clarke et al., 1987:48; Kelley and Micozzi 1984:385; Brothwell 1967:63).

The Genetic Resistance Theory

Due to this theoretical difficulty (Lahr and Bowman, 1992:652) and the devastating effects TB had on historic Natives, many archaeologists concluded that the Europeans were responsible for bringing TB to the New World. The devastating effects on the Natives could have been the result of their high genetic susceptibility to TB. Genetic resistance is built on natural selection and the Natives had a low level of such resistance because prior to Contact they had not been exposed to *M. Tuberculosis* (Dubos, 1965:173; Clarke et al., 1987:46; St. Hoyme, 1969:295). There was no exposure to several Old World pathogenic microorganisms because during the migration from Siberia approximately 10–40000 years ago (Sievers and Fisher, 1981:192), the "cold–screen" effectively filtered many infectious agents (St. Hoyme, 1969:295; Sievers and Fisher, 1981:195; Lahr and Bowman, 1992:652). However, I do not think TB crossed the land bridge in first place because there is no archaeological evidence for the

existence of TB in Old World prior to 10,000 B. P. For example, the earliest European skeleton is dated to 1000 B. P (Meachen, 1978:3), and the oldest Egyptian mummy with TB is dated to 3000 B.C. (Moller–Christensen, 1983:129; Dubos, 1965:232).

Although, I do not think that TB crossed the Bering Land Bridge, I also do not think that the Amerindians were genetically less resistant to TB. Instead, the devastation that was experienced by Natives of the New World was predominately due to unfavourable environmental changes, starvation, social disruption and general deprivation caused by the European settlers (Morse, 1961:503; Kunitz, 1983:68; Sievers and Fisher, 1981:196; Kelley and Eisenberg, 1987:90; Clarke et al., 1987:51). Furthermore, if genetic resistance is the only variable in immunity against TB, why do epidemics occur when the environment is degraded in Europe (Clarke et al., 1987:47)?

Accounting for the Absence of Cattle

A possible solution to the problem of the absence of cattle comes from the fact that the genus *Mycobacterium* has a diverse range of hosts: fish, reptiles, birds, soils and mammals (Cockburn 1963:219). The New World version of *M. Human* could have derived from opportunistic *Mycobacteria* (Klepinger 1982:203). These *Mycobacteria* could be from the soil, dogs, turkeys, llamas, wild birds, or perhaps even bison (Buikstra 1981:13, St. Hoyme, 1969:296; Cohen 1989:47; Wood et al., 1987:51; Lichtor and Lichtor 1957:1399). I am arguing then, that the New World prehistoric TB is not the same as the Old World TB because the Americas have their own flora and fauna. However, the variation of tuberculosis bacilli around the world is so small (Clarke et al., 1987:58) that the lesions found in the New World skeletons are "tuberculosis–like", or appear to be the same as the Old World tuberculosis.

The Spread of TB in the Pre-Contact New World

I believe that TB in the pre-Contact period was endemic in the New World (St. Hoyme, 1969:296; Pfeiffer, 1984:188; Clarke et al., 1987:51; Katzenberg, 1987:51–52). I conjecture that there is probably a high correlation between the advent of horticulture and TB. The relatively semi–permanent or permanent settlements associated with horticulture would result in frequent contact with bacteria and parasites associated with the accumulation of waste (St. Hoyme, 1969:300).

More importantly, horticulture groups and hunting and gathering groups experience infectious diseases in cycles (Pfeiffer, 1984:188). TB peaks as more vulnerable individuals are eliminated, the survivors become relatively resistant over a period of several generations and then the disease resumes a endemic pattern (Pfeiffer, 1984:188). TB would reactivate as a result of nutritional deficiency, forced migration, overpopulation, and warfare in the various populations (Steinbock 1987:55; Katzenberg 1987:51–52; Pfeiffer, 1984:188).

I think it is prudent not only to mention that TB was possible in small aggregate populations, but also to briefly mention the great cities of the New World. Clendinnen (1991:18), for example, describes early 16th century Tenochtitlan as a population "...tightly packed in extended or joint family compounds...to a density of perhaps thirteen thousand per square kilometer."

The huge Teotihuacan was another densely populated New World city. Teotihuacan was at the height of its power around 500 A.D.,

> ... and was larger than imperial Rome. For more than a half a millennium it was to Middle America what Rome, Benareres or Mecca have been to the Old World ...
>
> (Million 1973:82)

If Teotihuacan and Tenochtitlan were so similar to the cities of the Old World with regard to size, their dense population would be conducive to the spawning of TB. Further archaeological investigation is, however, necessary to confirm this hypothesis.

CONCLUSION

The diagnosis of TB is problematic in skeletal remains because there is considerable pathological overlap between infectious diseases, pyogenic osteomyelitis and blastomycosis having been seen to be easy to confuse with TB. Through the process of elimination, however, a sound argument can in most cases be made for the causative agent of a particular bone transformation. To date, I believe there is strong archaeological evidence suggesting that TB was present in the New World prior to Contact. The New World *M. Tuberculosis* was derived independently from the Old World because the former had its own reservoir of hosts to give rise to TB.

In analysing skeletal material it is important to remember that these individuals were once alive and breathing. I think that skeletal tuberculosis in an individual, especially in an egalitarian society, creates stress both economically and psychologically within the group and so it may diminish the cultural and economic development of a population (Fabrega, 1981:75). Thus, it is important to develop theories and analytical methods that deal with the distribution, or the extent in which a disease is present in a given population sample. In short, we must ask the question of whether the skeletal sample is representative of its past population.

As an aid to discovering the origin of *M. Tuberculosis* in the New World, Baker and Brothwell (1980) are very helpful in suggesting that archaeologists have to pay closer attention for possible abnormalities of bone in *animals*. I think that it would be equally fruitful to critically examine the archaeological evidence suggesting the prehistoric presence of leprosy in the New World. In the Old World, there appears to have been cross–immunity between TB and Leprosy.

Prior to 1492, there seems little doubt that some form of *M. Tuberculosis* was present in the New World. Was the form of *M. Tuberculosis* found in the New World exactly the same as that in the Old World? Was prehistoric TB precisely the same as the contemporary form of TB? Given the preceding evidence, and since *M. Tuberculosis* is subject to evolutionary change just like any other organism, I think it is very reasonable to answer in the negative to both of these questions.

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